VI F. Electroencephalogram in Man and Animals

All of the available information regarding the effects of carbon monoxide on the electroencephalogram relates to high concentration of the gas in the blood. In rabbits, levels of 45% and 74% carboxyhemoglobin were used respectively by Komura (1967) and De Valois and Schadé (1967). With these high concentrations there are various effects: shortening of the hippocampal electroencephalogram, appearance of slow waves in the neocortex, hypothalmus and caudate nucleus, and increase of the threshold of the arousal reaction in the neocortex. In rats, a decrease occurs in the amplitude and frequency of electrical cortical activity (Fodor et al, 1964).

The electroencephalogram of patients who become unconscious as a result of carbon monoxide poisoning shows the following changes: appearance of slow waves (Faure et al, 1965; Revol et al, 1966; Sasaki et al, 1966); low voltage fast waves (Tatetsu et al, 1968); delta waves (Mann, 1965; Tatetsu et al, 1967a); reduction in fast waves accompanied by rapid eye movement (Karacan et al, 1971); and a decrease in cortical evoked potential to photic, somatosensory and auditory stimulation (Kuroiwa et al, 1968). Most of these abnormalities remain even for two to five years after the acute exposure to carbon monoxide (Orioli and Cattania, 1965; Geier, 1966; Inanaga et al, 1966a and b; Revol et al, 1966; Sasaki et al, 1966; De Valois and Schadé, 1967; Tatetsu et al, 1967b; Harada and Kozuma, 1968; Tomonari, 1968; Grohme et al, 1969; Yasuoka, 1970; Harada et al, 1971; Totsuka et al, 1971).

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VII. OTHER ORGAN SYSTEMS

The investigations of the effects of carbon monoxide on organs other than the respiratory, circulatory and nervous systems are reviewed in this section. Two topics are remotely related to cigarette smoking and the coverage of each is confined to a bibliographic list: the effects of carbon monoxide on hepatic cells (see additional bibliographic list no. 12) and on unicellular organisms and lower vertebrates (see additional bibliographic list no. 13).

VII. A. Renal System

Some patients with severe carbon monoxide poisoning develop acute renal failure (Burck and Portwich, 1964; Nicolas and Nicolas, 1964; Kobulniczky and Koncz, 1966; Wijdeveld, 1968; Leonowicz, 1967; Linton et al., 1968; Quintana et al., 1969; Grosse and Neuhaus, 1970; Wojczuk and Chylak, 1971). The mechanism for the development of acute renal failure is believed to be other than hypoxia alone. Pauli et al. (1968a) noted a difference in the renal function of normal subjects at a high altitude (3,454 meters above sea level) for 10 days and that of 6 subjects exposed to carbon monoxide for 10 days with a carboxyhemoglobin concentration of 15%. In the presence of carboxyhemoglobin there is an increase of glomerular filtration rate with renal plasma flow remaining unchanged. This combination was interpreted to mean constriction of the efferent vessels with dilatation of the afferent arterioles. On the other hand, hypoxia caused an increase in renal plasma flow with an accompanying change in glomerular filtration rate in the same direction. In the same group of subjects, renal tubular electrolyte handling was consistent with the primary

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elevation of glomerular filtration rate during exposure to carboxyhemoglobin (Pauli et al., 1968b). There was no increase in urinary elimination of protein (Steiner et al., 1971).

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VII. OTHER ORGAN SYSTEMS

A. Renal System

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VII B. Pregnancy

Accidental carbon monoxide poisoning in a pregnant mother has been known to cause fetal death (Goldstein, 1965; Kells, 1969; Stokowski and Kęsiak, 1969). There is a report of a case without fetal abnormality (Larcan et al., 1970). Most other reports describe brain lesions in babies born of mothers who have suffered from carbon monoxide poisoning (Bankl and Jellinger, 1967; Kamraj-Mazurkiewicz, 1967; Szilágyi, 1967; Beaudoing et al., 1969; Matsuyama, 1969).

The diffusion of carbon monoxide across the placenta has been investigated in the sheep (Metcalfe et al., 1965; Longo et al., 1967, 1969; Longo, 1970; and Piédelièvre et al., 1969) and in humans (Gemzell et al., 1958; Friberg et al., 1959). Small quantities of carbon monoxide have been administered to pregnant women for measurement of the diffusing capacity of the placenta (Delivoria-Papadopoulos and Coburn, 1972). The radioactive form of carbon monoxide administered by inhalation has been used to localize the placenta in a mother close to parturition (Glass et al., 1968; Hakim, 1970).

Carboxyhemoglobin levels in maternal and fetal blood have been measured by several investigators (Table 7). The levels in mothers who smoked were higher than in those who were nonsmokers. **1005051166**

Respiration in infants of mothers who smoked took longer to establish than in those of nonsmokers (Heron, 1962; Tanaka, 1967; Scoppetta, 1968). There was a higher incidence of prematurity among mothers who smoked (Girond, 1967) and a higher neonatal mortality rate (Comstock et al., 1971). The period of gestation is 29 to 34 hours

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shorter for smokers than for nonsmokers (Buncher, 1969). The birth weight of babies born of mothers who smoked was lower (Mantell, 1964; Baribaud et al., 1970; Murphy and Mulcahy, 1971; Astrup et al., 1972).

There is a rise in carboxyhemoglobin level in the blood of the newborn. Behrman et al. (1971) have noted a mean rise of 6.98% in 16 newborn infants, which related to the level of pollution in the nursery. When the concentration of carbon monoxide exceeded 20 ppm, there was a decrease of 11.4% in the oxygen-carrying capacity during the first 24 hours and a decrease of 13.9% during the 25 to 85 hours after birth. These changes do not correlate with the history of maternal smoking and are caused by the concentration of carbon monoxide in the atmosphere.

The effect of carbon monoxide alone on the birth weight of rabbits has been investigated by Astrup et al. (1972) and in rats by Younoszai et al (1969). An exposure to 90 ppm carbon monoxide resulting in 9 to 10% carboxyhemoglobin in the blood caused a reduction in birth weight and an increase in neonatal mortality. The influence of a level of 5% carboxyhemoglobin was not investigated.

(Table 7 appears on the following page.)

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Table 7. Carboxyhemoglobin levels of fetal and maternal blood.

Reference (Year)	Nonsmoking mothers			Smoking mothers		
	No of mothers	Maternal Mean ± SD (Range)	Fetal Mean ± SD (Range)	No of mothers	Maternal Mean ±SD (Range)	Fetal Mean ± SD (Range)
Haddon, Nesbitt and Garcia (1961)	7	(0.1-3.1)	(0.2-2.8)	12	(0.1-8.4)	(0.1-9.8)
Heron (1962)	27	2.6 (0.4-4.4)	2.5 (0.2-3.6)	21	6.7 (1.6-14)	5.0 (1.1-9.2)
Young and Pugh (1963)	9	1.6 ± 0.42	1.61 ± 0.37	6	2.0 ± 0.77	2.4 ± 0.74
Bjure and Fällström (1963)	8	1.0 (0.7-1.43)	1.15 (0.77-1.64)			
Younaszai, Kacic and Haworth (1968)	11	1.2 (0-3)	0.> (0-1.5)	10	8.3 (2-12)	7.3 (2-10)
Cole, Hawkins and Roberts (1972)	129	1.2 (0-2.4)	2.2	93	4.1 (0.5-14)	7.5
Astrup, Trolle, Olsen and Kieldsen (1972)	884	0.87		824	1.92	

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VII. OTHER ORGAN SYSTEMS

B. Pregnancy

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VII C. Endocrine System

The sex organs in animals influence the survival rate in carbon monoxide exposure. Stupfel et al. (1971) noted a higher mortality in male than in female mice and that castration increased the mortality in the female. The mechanism by which estrogens and progestogens increase the tolerance to carbon monoxide poisoning is not known. On the other hand, carbon monoxide in vitro depresses some of the enzymes that are involved in the biosynthesis of estrogen (Meigs and Ryan, 1971). The structure of the ovary was unaffected and all the stages in the development of the ova were noted in rats exposed to carbon monoxide in vivo (Mamatsashvili, 1970). The effects of carbon monoxide on male fertility are uncertain (Wittgens, 1966).

Adrenalectomy increased the mortality of rats and mice in response to carbon monoxide (Pukhov, 1964). In animals with adrenals intact, adrenal cortical activity is stimulated by exposure to carbon monoxide (Katsuki et al., 1966; Hanke and Kieres, 1967). Stimulation of the adrenal medulla also occurred, which contributed to the hyperglycemia (Bour et al., 1968; Hirano et al., 1968). There are substances in the rat adrenal gland which combine with carbon monoxide (Harding et al., 1964; Wilson and Harding, 1970). The role of these substances in mediating the stimulation of the adrenal gland and in reducing the lethality of carbon monoxide has not been considered. The reduction in rate of growth of rats exposed to carbon monoxide is associated with a decrease in activity of the thyroid gland (Truhaut et al., 1965; Pogrund, 1969).

The inhalation of carbon monoxide in human subjects causes an

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elevation of blood sugar (Hirano et al, 1967; Nakao, 1969). The hyperglycemia induced by cigarette smoking is believed to be due to nicotine stimulation of the adrenal medulla (Haggard and Greenberg, 1934). The contribution of carboxyhemeglobin derived from cigarette smoking has not been estimated.

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VII. OTHER ORGAN SYSTEM

C. Endocrine System

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